cells was evaluated in a cell proliferation assay. NF- κ B intracellular levels were determined by Western Blot. TNF- α production was measured in culture medium supernatants by ELISA.

Results: An enhancement in cell proliferation was found in CS+GLU treatments at a concentrations of 100 and 200 μ g/ml, increasing 1,60-fold ($p<0.01$) and 2,04-fold ($p<0.001$), respectively, compared to untreated cells. In addition, myoblasts were then incubated with IL-6 (50 ng/ml) for 72 hour in order to induce an inflammatory environment. The results showed an IL-6 induced-reduction on cell proliferation in all groups, although the data did not reach statistical significance. Therefore, an IL-6 inhibitory effect on cell proliferation in human muscle cannot be ensured. We also measured the effect of the combined treatment CS+GLU on NF- κ B activation and TNF- α production in human skeletal muscle cells in primary culture. Despite of TNF- α levels were undetectable in cell supernatants, preliminary data showed a slight reduction on NF- κ B signalling pathway. Global gene expression profiles, measured by microarrays and GeneChip Human Gene 1.0 ST Arrays (Affymetrix), will also be analysed.

Conclusions: The mechanisms of action involved in the potential therapeutic effect described in an *in vivo* injured muscle model seem to be related with an increase in muscle cell proliferation, together with blocking NF- κ B nuclear translocation and TNF α production. Although further investigation is required, these pre-clinical data suggests potentially positive effects of CS and GLU administration for the treatment of skeletal muscle injuries in sports medicine.

Disclosure of Interest: None declared

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AB0061

SERUM IL-37 AS AN EFFICIENT BIOMARKER OF DISEASE ACTIVITY IN PATIENTS WITH ANKYLOSING SPONDYLITIS: POST-HOC ANALYSIS OF PLANETAS STUDY

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Background: IL-37 is an anti-inflammatory cytokine which belongs to IL-1 cytokine superfamily. Patients with ankylosing spondylitis (AS) also had a higher serum IL-37 concentration than controls, and were also associated with other inflammatory markers such as CRP, ESR, and BASDAI levels.¹ However, the relationship between serum IL-37 and disease activity after anti-TNF treatment has not been reported.

Objectives: To investigate the association of serum IL-37 level with disease activity in AS patients who were treated with infliximab.

Methods: Patients were recruited from the PLANETAS study (NCT01220518). Patients with active AS were treated with CT-P13 or infliximab originator. The serum levels of IL-37 were measured at week 0 and week 30 with specific ELISA. Other demographic, laboratory and clinical variables were evaluated simultaneously.

Results: Fifty patients with active AS (BASDAI >4) were analysed. The median age of patients was 40 years old [Interquartile range (IQR), 33.8–49.5], and the median BASDAI score was 6.7 (IQR 5.3–7.9). Compared to baseline, all measured clinical and laboratory parameters and serum IL-37 were significantly reduced. There was a statistically significant correlation between IL-37 and CRP, BASDAI at baseline, but not at week 30. Additionally, the differences (Δ) of parameters at baseline and at week 30 were assessed on their associations with other disease activity parameters. Delta IL-37 was significantly correlated with Δ CRP, Δ BASFI and Δ BASDAI. The ROC curves of serum biomarkers for ASAS 20 achievement revealed that AUC value for Δ IL-37 (AUC=0.74) was similar with Δ ESR (AUC=0.71, $p=0.64$) and superior than Δ CRP (AUC=0.54, $p<0.01$). AUC value for Δ IL-37 of ASAS 40 achievement was 0.66, which was similar with Δ ESR (AUC=0.60, $p=0.38$) and superior than Δ CRP (0.46, $p<0.01$).

Abstract AB0061 – Table 1. Demographic & Clinical characteristics of analysed patients, comparing at baseline and at week 30.

Variables	At baseline	At week 30	P
Age	40.0 (33.8–49.5)	-	N/A
Female sex	7 (14%)	-	N/A
IL-37	105.1 (25.9–286.9)	44.5 (18.2–153.9)	<0.01
ESR	35.0 (28.0–41.3)	7.5 (5.0–16.0)	<0.01
CRP	158.1 (53.4–291.4)	20.0 (7.4–73.1)	<0.01
BASDAI	6.7 (5.3–7.9)	3.3 (1.9–4.5)	<0.01
BASMI	4.0 (3.0–5.0)	3.0 (2.0–4.0)	<0.01
BASFI	6.6 (5.3–8.1)	3.3 (1.6–5.9)	<0.01
PGD	70.5 (57.3–80.8)	33.0 (14.8–53.3)	<0.01

Abstract AB0061 – Table 2. Correlations between IL-37 and other disease activity markers; at baseline and the differences between baseline and at week 30.

	At baseline			Difference between baseline and week 30		
	IL-37	ESR	CRP	Δ IL-37	Δ ESR	Δ CRP
IL-37		0.14	0.39**	Δ IL-37	0.24	0.36*
ESR	0.14		0.57**	Δ ESR		0.50**
CRP	0.39**	0.57**		Δ CRP	0.36*	0.50**
BASDAI	0.31*	0.31*	0.23	Δ BASDAI	0.32*	0.25
BASMI	0.05	0.09	0.16	Δ BASMI	-0.05	0.06
BASFI	0.20	0.36**	0.25	Δ BASFI	0.34*	0.35*
PGD	0.25	0.10	0.06	Δ PGD	0.16	0.28
						-0.06

Abstract AB0061 – Figure 1. Figure 1 ROC Curves of disease activity markers of ASAS 20 and ASAS 40 achievement.

Conclusions: Serum IL-37 levels are associated with BASDAI, patient's pain score, and CRP in active AS patients. Change in serum IL-37 level may serve as an efficient biomarker predicting the improvement of BASDAI and the achievement of ASAS 20 and 40 after anti-TNF treatment in AS patients.

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AB0062

RECIPROCAL INTERACTION BETWEEN MACROPHAGE MIGRATION INHIBITORY FACTOR AND INTERLEUKIN-8 IN GOUT

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Background: Macrophage migration inhibitory factor is a proinflammatory, chemotactic, and tissue destructive cytokine.

Objectives: This study determined monosodium urate crystal-induced macrophage migration inhibitory factor production and its interaction with interleukin-8 in gout.

Methods: Peripheral blood, synovial fluid, and clinical data were obtained from 98 patients with gout. Synovial fluid and serum concentrations of macrophage migration inhibitory factor and interleukin-8 were measured. Synovial fluid monocytes and neutrophils were cultured with monosodium urate crystals and the cytokine production was determined. The signalling pathways involved were determined using signal inhibitors. The interaction between macrophage migration inhibitory factor and interleukin-8 was investigated.

Results: Synovial fluid macrophage migration inhibitory factor was higher in acute gout and that in serum was higher in patients with intercritical gout compared with controls. Synovial fluid macrophage migration inhibitory factor was positively correlated with synovial fluid leukocyte and neutrophil counts and interleukin-8. The expression of macrophage migration inhibitory factor was similar in synovial fluid neutrophils and monocytes, while interleukin-8 was higher in monocytes. Monosodium urate crystals induced macrophage migration inhibitory factor production in monocytes and interleukin-8 production in neutrophils. This effect was decreased by inhibiting Fc-gamma receptor 1 and toll-like receptor 4. Interleukin-8 increased macrophage migration inhibitory factor production in monocytes while macrophage migration inhibitory factor increased interleukin-8 production in neutrophils.